CARDIOVASCULAR SYNDROMES

Minsk BSMU 2022

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА ПРОПЕДЕВТИКИ ВНУТРЕННИХ БОЛЕЗНЕЙ

КЛИНИЧЕСКИЕ СИНДРОМЫ ПРИ ЗАБОЛЕВАНИЯХ СЕРДЕЧНО-СОСУДИСТОЙ СИСТЕМЫ

CARDIOVASCULAR SYNDROMES

Учебно-методическое пособие

Минск БГМУ 2022

УДК 616.24-008.6(075.8)-054.6
ББК 54.101я73
К49

Рекомендовано Научно-методическим советом университета в качестве учебно-методического пособия 18.05.2022 г., протокол № 5

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Доценко, Э. А.

К49 Клинические синдромы при заболеваниях сердечно-сосудистой системы = Cardiovascular syndromes : учебно-методическое пособие / Э. А. Доценко [и др.]. – Минск : БГМУ, 2022. – 27 с.

ISBN 978-985-21-1141-6.

Содержит сведения о клинических синдромах при заболеваниях сердечно-сосудистой системы.

Предназначено для студентов 3-го курса медицинского факультета иностранных учащихся, обучающихся на английском языке по специальности «Лечебное дело».

УДК 616.24-008.6(075.8)-054.6
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ISBN 978-985-21-1141-6

EXPLANATORY NOTE

Total duration of classes is 3,5 hours.

Diseases of the cardiovascular system have a leading place in the mortality structure worldwide. The clinical diagnosis in case of cardiovascular pathology is based on a study of: the patient's complaints; present and past medical history (*anamnesis of morbi and vitae*); objective examination data (inspection, palpation, percussion, auscultation); the results of laboratory and instrumental methods. The individual manifestations of the disease and the mechanisms of symptom occurrence also have great value.

Semiotics is the science studied symptoms and syndromes of internal organs diseases. The basis of clinical diagnosis is consisted of five classical methods of physical examination methods: interview, inspection, palpation, percussion and auscultation. However, without a good knowledge of pathological symptoms and syndromes, it is impossible to fully master the basics of clinical diagnosis. The proposed teaching aid outlines the main clinical syndromes in cardiovascular diseases. The knowledge of these syndromes will further help to master the clinical diagnosis of cardiovascular diseases.

The purpose of the class: to teach students the most common symptoms and syndromes of cardiovascular disease; to teach methods of diagnostics of such syndromes and clinical value of them.

Objectives of the class:

1. To study the etiology and pathogenesis of the main clinical syndromes in cardiovascular diseases.
2. Master the subjective, objective, laboratory and instrumental methods of examination in case of the main cardiovascular syndromes.
3. To consolidate theoretical knowledge on the main clinical cardiovascular syndromes by examining patients with cardiac diseases.

Pathologies of discussion:

1. Arterial hypertension syndrome. Clinical signs.
2. Peripheral circulation disorders syndrome. Clinical signs.
3. Acute coronary syndrome. Clinical signs.
4. Syndrome of acute left ventricular failure. Clinical signs.
5. Chronic heart failure syndrome. Clinical signs.
6. Arrhythmias (Heart Rhythm Disorders). Clinical signs.
7. Acute circulatory failure. Clinical signs.

ARTERIAL HYPERTENSION SYNDROME

Arterial hypertension is defined as increasing arterial blood pressure (BP)
excess of 140 mm Hg systolic one (SBP),
and/or excess of 90 mm Hg diastolic blood pressure (DBP).

Elevated BP is the leading global contributor to premature death due to cardiovascular events (hemorrhagic stroke, ischemic stroke, myocardial infarction, sudden death, heart failure, peripheral artery disease, as well as end-stage renal disease).

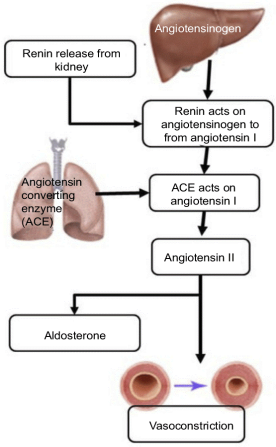


Fig. 1. Activation Renin-Angiotensin-Aldosterone System (RAAS)

Hypertension occurs as two major types:

1. Essential or idiopathic (cause unknown) hypertension, the most common (95 %)
2. Secondary hypertension, which results from kidney disease, endocrine disorders or another identifiable cause.

Mechanisms (pathogenesis) of Arterial Hypertension:

1. Activation of sympathoadrenal system.
2. Activation of Renin-Angiotensin-Aldosterone System (RAAS) (Fig. 1).
3. Insulin resistance and/or hyperinsulinemia
4. Deficiency of kallikrein system
5. Deficiency of neutral lipid and a prostaglandin produced in renal medulla.
6. Dysfunction of endothelial cells.

Risk factors of Arterial Hypertension are divided in modifiable and non-modifiable (Table 1).

Table 1

Risk factors of Hypertension

Non-modified	Modified
– Age – Genetics and family history – Sex (male) – Family and personal history of hyperlipidemia – Family and personal history of diabetes – Race	– Cigarette smoking, alcohol – Stress – Sedentary lifestyle – Weight (obesity and metabolic syndrome) – Dietary habits (high alcohol intake, high sodium intake, low potassium intake)

According to the WHO classification, arterial hypertension has 3 grades (table 2). BP category is defined according to seated clinic BP and by the highest level of BP, whether systolic or diastolic.

Table 2

Arterial hypertension classification according to level of BP (WHO)

Category	SBP, mmHg		DBP, mmHg
Optimal	< 120	and	< 80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥ 180	and/or	≥ 110
Isolated systolic hypertension	≥ 140	and	< 90

Systolic hypertension in the elderly: The age-related rise in systolic BP was considered part of the «normal» aging process, and isolated systolic hypertension (ISH) in the elderly was largely ignored. However, evidence from 3 major studies indicate without a doubt, that benefits of treating are even greater than treating moderate hypertension in middle-aged patients.

«Malignant» hypertension: This refers to severe hypertension (e. g. systolic > 200, diastolic >130 mmHg) in conjunction with bilateral retinal hemorrhages and exudates; papilledema may or may not be present. Symptoms are common e. g. headache ± visual disturbances which require urgent treatment. However, it may precipitate acute renal failure, heart failure, or encephalopathy which are hypertensive emergencies. Untreated, 90 % die in 1 year; treated, 70 %: survive 5 years.

Complaints:

– **Cerebral complaints**: headache, dizziness, vision disorders, buzzing in the ears and head, irritation (due to disorders of vessel tone, their widening is changed spasm. It results in disorders of cerebral circulation. There are also inflation of the cerebral vessels by increased BP).

– **Cardiac complaints**: heart pain, palpitation and interruption of the heart beat.

– **General complaints**: fatigue, sleep disorders, decreasing work productivity .

Objective examination of a patient in case of Arterial hypertension can include following points (table 3).

Table 3

Objective examination in case of Hypertension

Method	Result
Visual examination	flush of the face and sclera (erythema, reddish skin, red eyes)
Palpation	Pulse is hard, intense. Apical impulse is displaced to the left, resistant; it has high amplitude
Percussion	The left border of relative heart dullness is displaced to the left due to left ventricle hypertrophy.
Auscultation	S1 is diminished on the apex (muscle component) S2 is accented on the aorta (high pressure)

End the tabl. 3

Method	Result
BP	Serial blood pressure measurements greater than 140/90 mm Hg confirm hypertension. 24-hour ambulatory blood pressure monitoring: average BP greater than 130/80 mm Hg confirms hypertension.
Laboratory and instrumental examinations are done not for hypertension diagnosis, but for primary diseases or complication detection.	
Laboratory examination	***Biochemical blood test***: – **Serum potassium**: Levels less than 3.5 mmol/L may indicate adrenal dysfunction (primary hyperaldosteronism). – **Serum urea creatinine levels**: urea and creatinine levels can be normal. Urea level elevated more than 8,5 mmol/L and creatinine levels elevated more than 115 mmol/L suggest kidney disease. ***Urinalysis***: The presence of protein, red blood cells, and white blood cells may indicate glomerulonephritis.
Instrumental examination	– **Electrocardiography** may show left ventricular hypertrophy or ischemia (depressed ST segment and negative T-wave in I, II, AVL, V4-6). – **Chest X-ray** may show cardiomegaly. – **Echocardiography** may show left ventricular hypertrophy. There may also thickened left ventricular walls and/or thickened inter-ventricular septum. – **Ophthalmoscopy** reveals arteriovenous nicking and in hypertensive encephalopathy, papilledema may be present – **Renal arteriography** may show renal artery stenosis

Classification and clinical presentation of the essential hypertension

I stage — episodic elevation of BP with cerebral, cardiac and minor symptoms without any other signs except high BP.

II stage: Permanent symptoms and signs of target organs symptoms without their failure:

– Heart — left ventricle hypertrophy (apical impulse, left heart border, ECG, Echocardiography, X-Ray);

– Eyes — hypertensive retinopathy I–II;

– Kidney — proteinuria, increased blood creatinine.

III stage — Permanent symptoms and signs of the target organ failure (complicated stage)

Heart — myocardial infarction, heart failure.

– Brain — cerebrovascular accident, chronic hypertensive encephalopathy and vascular dementia;

– Eyes — hypertensive retinopathy III–IV;

– Kidney — proteinuria, increased blood creatinine, chronic renal failure;

– Vessels — aortic dissecting aneurysm.

PERIPHERAL CIRCULATION DISORDERS

Peripheral Arterial Disease Syndrome

Peripheral arterial disease is a chronic disease in which atherosclerotic plaque builds up in the arteries to the legs (fig. 2). This buildup typically occurs gradually. The blood flow in that artery become limited or blocked all together. The causes of peripheral arterial disease include smoking, high cholesterol level, high blood pressure, diabetes, and obesity. Genetic factors also play a certain role.

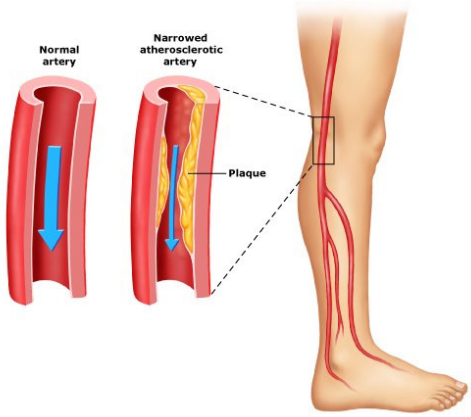


Fig. 2. Peripheral arterial diseases

Complaints:

Fatigue or cramping of the muscles (claudication) in the leg, thigh, hip, or buttock are usual. Typically, the discomfort is felt after certain physical activities (such as walking or climbing stairs) and goes away with rest. Other complaints may be leg numbness or weakness, coldness in lower leg or foot, sores on the toes, feet or legs that won't heal, and erectile dysfunction in men.

Objective examination of a patient in case of Peripheral arterial diseases can include following points (table 4).

Table 4

Objective examination in case of Peripheral Arterial Disease

Method	Result
Visual examination	Change in the color of leg's skin (pallor) Hair loss on feet and legs
Palpation	Cold extremity No pulse or a weak pulse in the legs or feet
Percussion	–

End the tabl. 4

Method	Result
Auscultation	–
Laboratory examination	Biochemical blood test: – Total cholesterol level more than 5,2 mmol/L, – Low-density lipoprotein level more than 3,5 mmol/L, – Triglyceride level more than 2 mmol/L indicate high risk of atherosclerotic process
Instrumental examination	**Ankle brachial index**. The test involves taking a blood pressure reading at the ankle and comparing it to that in the arm (normal range 1,0–1,4. In case of peripheral arterial diseases this index decreases less than 0,99). **Vascular ultrasound** can show narrowed blood vessels, atherosclerotic plaque, blood clots, and areas where the blood flow is blocked. **An angiogram** detects blockages of arteries using X-rays taken during the injection of a contrast agent (iodine dye)

Peripheral Venous Disease Syndrome

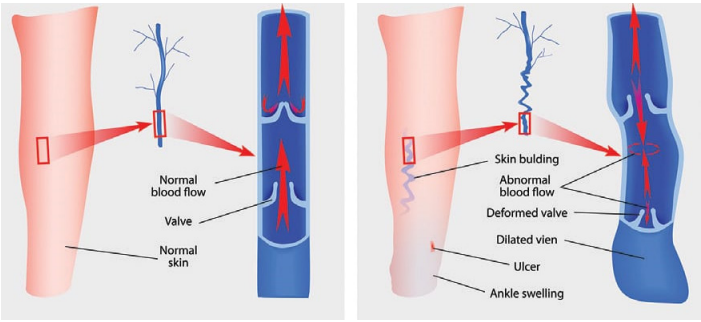
The most common cause of peripheral venous disease is a blood clot that blocks a vein. A clot forms when vein walls become weak and blood flow slows. When the clot is in a vein deep within the body, it is called deep vein thrombosis. When the clot is in a vein closer to the skin, it is called superficial thrombophlebitis.

Venous thrombosis may be caused by:

1. Blood stasis:
– arrhythmia;
– heart failure;
– immobility for a long period
– varicose veins (Fig. 3);
– obesity.

2. Damage to blood vessels:
– trauma;
– bone fracture.

3. Coagulation disorders:
– cancer;
– pregnancy;
– certain medicines (for example, contraceptives)
– polycythemia.



a ***b***

Fig. 3. Peripheral Venous Disease formation:
a — Normal vein; *b* — Varicose vein

Complaints:

Patients can complain to varicose veins, leg edema, itching, cramping, heaviness in the legs and, in later stage, to open skin sores (ulcers).

Objective examination of a patient in case of Peripheral venous diseases can include following points (table 5).

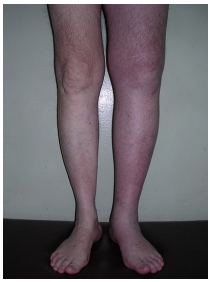
Table 5

Objective examination in case of Peripheral Venous Disease

Method	Result
Visual examination	Change in the color of leg skin (cyanosis) Leg edema (usually unilateral)
Palpation	Leg is painful in palpation. Firm vein can be palpable. Skin temperature is warm
Percussion	–
Auscultation	–
Laboratory examination	– *Coagulation blood test*: Prothrombin time and INR may be decreased (indicates increased coagulation) – *Complete blood count*: Thrombocytosis indicates blood diseases
Instrumental examination	**Vascular ultrasound** can show dilated veins, blood clots, and areas where the blood flow is blocked

Superficial Thrombophlebitis Syndrome

Superficial thrombophlebitis is an inflammatory disorder of superficial veins which coexist with venous thrombosis (fig. 4). Superficial thrombophlebitis has all signs of inflammation. Vascular endothelial injury results in thrombus formation by triggering an inflammatory response that results in immediate platelet adhesion. When blood flow is abnormal due to turbulence or stasis, vessel wall become involved in the process, micro-thrombi could increase and subsequently form macroscopic thrombi.



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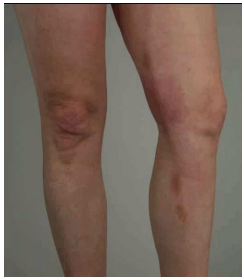


b

Fig. 4. Superficial thrombophlebitis:
a — edema, skin color changed; *b* — inflamed vein is visible

Complaints:

Acute superficial thrombophlebitis begins suddenly, body temperature reaches 38–39 °C, occurs with chills. Pain in the damaged area is severe, infiltration is along the affected vein, regional lymph nodes are enlarged, tissue swelling is visible along the affected vein. Volume of the leg or thigh is increased due to swelling.

Objective examination of a patient in case of Superficial thrombophlebitis can include following points (table 6).

Table 6

Objective examination in case of Superficial thrombophlebitis

Method	Result
Visual examination	Change in the color of leg skin (erythema) Leg edema (usually unilateral)
Palpation	Leg is extremely painful in palpation. Firm vein can be palpable. Skin temperature is hot
Percussion	–
Auscultation	–
Laboratory examination	– *Coagulation blood test*: Prothrombin time and INR may be decreased (indicates increased coagulation) – *Complete blood count*: leukocytosis is the result of inflammation
Instrumental examination	**Vascular ultrasound** can show dilated veins, blood clots, and areas where the blood flow is blocked

Lymphedema Syndrome

Lymphedema is swelling due to build-up of lymph fluid in the body. If the lymph drainage is affected, the fluid cannot drain. It usually happens in the arms or legs. This swelling develops slowly over several months.

Lymphedema develops, as a rule, on the back of the foot or in the lower leg (Fig. 5). This edema increases in summer, after a physical activity; reduces with cold weather (autumn, winter) and after a long rest. The skin has a normal color, and normal temperature. The lymphedema has firm consistency, it is painless. The skin fold is collected with difficulty, there are no dimple on it when pressed.

Complaints:

Patients can complain for swelling of part or all of the arm or leg, heaviness or tightness in affected extremity, and thickening of the skin.

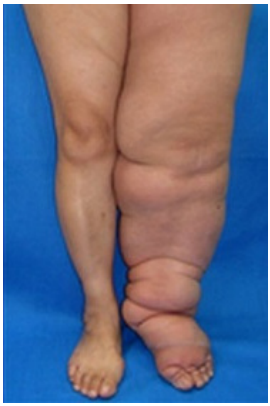


Fig. 5. Lymphedema

Objective examination of a patient in case of Lymphedema can include following points (table 7).

Table 7

Objective examination in case of Lymphedema

Method	Result
Visual examination	Skin has a normal color Feet and leg edema (unilateral or bilateral) presence
Palpation	Palpation is painless Skin temperature is normal No dimple appears after pressure to the skin
Percussion	–
Auscultation	–
Laboratory examination	–
Instrumental examination	**Ultrasound** sometimes can show increased lymph nodules

ACUTE CORONARY SYNDROME

The term «Acute coronary syndrome» (ACSs) is applied to patients in whom there is a suspicion of acute myocardial ischemia. ACS is related with decreased blood flow in the coronary arteries such that part of the myocardium is unable to function properly or dies. Acute coronary syndrome usually results from atherosclerosis, when cholesterol plaques build up in the walls of coronary arteries. When a plaque deposit ruptures or splits, a blood clot forms. This clot blocks the flow of blood to heart muscles (fig. 6).

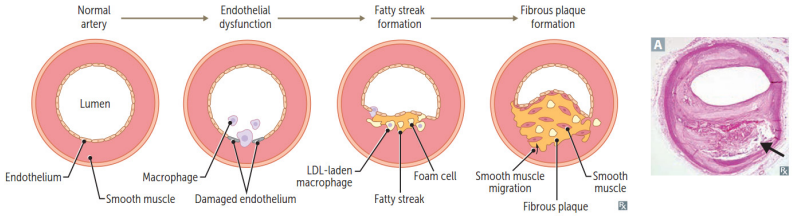


Fig. 6. Schematic representation of sequential progression of coronary artery lesion morphology, beginning with stable chronic plaque responsible for typical angina and leading to the various acute coronary syndromes

Overview: Coronary artery obstruction or rupture can result in a variety of ischemic condition which fall under the term of acute coronary syndrome. Plaque rupture, thrombosis and spasm of coronary artery are considered substrate for ACS.

Types of Acute coronary syndrome are as follows:

– Unstable angina;
– ST-elevation myocardial infarction (STEMI);
– Non-ST-elevation myocardial infarction (NSTEMI).

Definitions

• **Unstable angina** is defined by the absence of biochemical evidence of myocardial damage. It is characterized by specific clinical findings of prolonged (> 20 min) angina at rest; new onset of severe angina; angina that is increasing in frequency, longer in duration, or lower in threshold; or angina that occurs after myocardial infarction.

• **Acute myocardial infarction**: Death of myocardial tissue because of inadequate blood flow.

- ST- segment elevation myocardial infarction (STEMI): MI as defined as in acute myocardial infarction, with ST-segment elevation more than 0.1 mV in two or more contiguous leads, and elevated cardiac biomarkers.

- Non-ST segment elevation myocardial infarction (NSTEMI): MI, but without ST-segment elevation. May have other ECG changes, such as ST-segment depression or T-wave inversion. Which will have elevated cardiac biomarkers.

All three syndromes occur when a vulnerable plaque ruptures, leading to platelet activation and aggregation, resulting in the formation of intra-coronary thrombus.

Risk factors of acute coronary syndrome are as follows (table 8).

Table 8

Risk factors of Acute coronary syndrome

Non-modified	Modified
– Age – Family history – Male sex – Race	– Smoking – Hypertension – Dyslipidemia – Diabetes mellitus – Obesity, high calorie intake diet – Physical inactivity (sedentary lifestyle) – Psychological stress

Complaints:

1. Retrosternal Pain in case of Acute coronary syndrome has the following characteristics:

– Duration at least 30 min;
– Radiation to the neck, arms, jaw, epigastrium or back (left scapular area);
– The character (type) of pain is described as crushing, heaviness or squeezing like a tight band. Patients classically clinch their fist and hold it on their chest to describe the pain (Levine's Sign) (fig.7);

– Worse with physical or emotional exertion;
– Not relieved by rest;
– Nitrate spray (within a couple of minutes) may not always relieve the pain:

2. Anxiety and fear of impending death.
3. Nausea and vomiting.
4. Breathlessness.
5. Collapse/syncope.

Fig. 7. Levine's Sign

Table 9

Objective examination in case of Acute coronary syndrome

Method	Result
Visual examination	Skin has a pale color, sweating Extremities are cold Low-grade fever develops after several days (resorption of necrotic tissue)
Palpation	Pulse: tachycardia or bradycardia, arrhythmia. Hypotension, low pulse pressure
Percussion	Heart borders may be displaced according previous condition (Hypertension, Left ventricle hypertrophy)
Auscultation	Heart sounds are weak, especially S1 at the apex, Gallop rhythm (third heart sound) Bilateral lower-lobes lung crepitation due to acute heart failure (lung congestion)
Laboratory examination	*CBC*: Leukocytosis, increased Erythrocytes sedimentation rate *Biochemical blood test*: Cardiac Enzymes (Leakage of protein from injured cardiac myocytes): – *Troponin* rises in 6 hours, peaks in 1–2 days, lasts 2 weeks. It has high sensitivity and specificity. – *Creatinine Kinase MB-fraction* (CK-MB) rises in 4–6 hours, peaks at 12 hours and at 2 days drops off. – Myoglobin rises in 2 hours. It has high sensitivity, but low specificity. *Markers of atherosclerotic process*: – Total cholesterol level more than 5,2 mmol/L – Low-density lipoprotein level more than 3,5 mmol/L *ECG*: – ST segment elevation or depression, – pathological Q wave, – T wave flattening or negative
Instrumental examination	Echocardiography shows regional heart wall motion abnormalities Coronary angiography

Differential diagnosis between STEMI and NSTEMI is done according special algorithm (fig. 8).

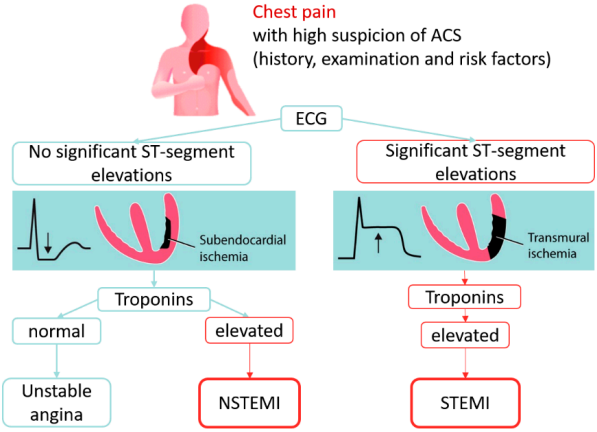


Fig. 8. Algorithm of Acute coronary syndrome diagnosis

SYNDROME OF ACUTE LEFT VENTRICULAR FAILURE

Acute left ventricle heart failure occurs when the left ventricle muscle is suddenly weakened. In such a case, the heart is unable to pump oxygen-rich blood from the lungs through the body.

Stages:

– Cardiac asthma (fluid in the interstitial tissue);
– Pulmonary edema (fluid in the alveoli) (fig. 9).

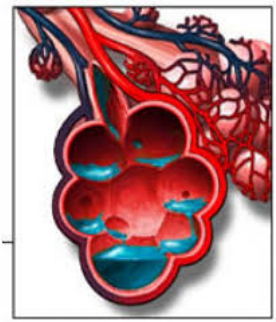


Fig. 9. Pulmonary edema (fluid inside of alveoli)

High hydrostatic pressure in the lung capillaries leads to fluid movement from the capillaries into the interstitial space. It happens at stage of cardiac asthma. Then fluid goes into the lumen of the alveoli and pulmonary edema develops.

Normal pressure in the lung capillaries is 2–10 mm Hg. Cardiac causes of an increase pressure are the following:

– increased diastolic pressure in the left ventricle (ischemic heart diseases, myocardial infarction, valvular heart diseases, hypertensive crisis);

– high load on the myocardium (thyrotoxicosis, anemia, arrhythmias, intravenous infusions of large fluid volume, constrictive pericarditis);

– increased pressure in the left atrium: mitral stenosis, myxomas of the left atrium.

Acute left ventricular failure complains and physical examination are following (table 10).

Table 10

Complains and objective examination in case of acute left ventricular failure

Method	Result	
	Cardiac asthma	**Pulmonary edema**
Complaints	Shortness of breath (dyspnea) Orthopnea Chest pain (tightness) Wheezing or gasping	
	Cough (dry)	Cough (with white or pink foamy sputum)
Visual examination	Pale skin or cyanosis, tachypnea	
Palpation	Pulse is weak or/and irregular, tachycardia	
Percussion	Relative heart dullness left border is shifted to the left	
Auscultation	Heart sounds weaken, S3 at the apex (gallop rhythm), murmurs S2 louder at pulmonary artery Weaken vesicular breathing	
	Dry rales (wheezing)	Wet rales (crackles), crepitation
Laboratory examination	Biochemical blood test shows increased level of BNP (brain natriuretic peptide)	
Instrumental examination	ECG — left ventricle hypertrophy, arrhythmia, conduction block, non-specific ST/T wave changes Pulse oximetry — low SpO_2 Echocardiogram — abnormal heart valves, low myocardial contractility (↓EF), high pressure in Pulmonary artery	
	Chest X-ray — congestion, pleural effusion, increased cardio-thoracic ratio	
	Cephalization of the lung vasculature is presence of symmetrical homogeneous shadows in the lung roots («bat wing»)	Bilateral multiple diffuse shadows of varying intensity

CHRONIC HEART FAILURE SYNDROME

Heart failure is a condition in which the heart muscle is unable to pump enough blood to meet the body's needs for blood and oxygen. Basically, the heart can't keep up with its workload.

Chronic heart failure is a clinical syndrome characterized by *typical symptoms* (e. g. breathlessness, ankle swelling and fatigue) that may be accompanied by signs (e. g. elevated jugular venous pressure, pulmonary crackles and peripheral edema) caused by a structural and/or functional cardiac abnormality, resulting in a reduced cardiac output and/or elevated intracardiac pressures at rest or during stress.

Heart failure etiology are as follows:

– Myocardial dysfunction: coronary heart disease, myocarditis;
– Pressure overload (afterload): hypertension, valvular stenosis;
– Volume overload (preload): valvular regurgitation.

Heart Failure Classification include *New York Heart Association Functional Classification* (NYHA) and Stages (Russian) (table 11).

Table 11

Heart Failure Classification

NYHA	Characteristics	Stage (Russian)	Characteristics
I	No symptoms at rest. Intensive physical exertion leads to SOBOE*	1	No symptoms at rest. Intensive physical exertion leads to SOBOE
II	Moderate physical exertion leads to SOBOE, fatigue, palpitation	II A	Congestion in one circle of blood circulation (pulmonary OR systemic)
III	Minimal physical exertion leads to SOBOE, fatigue, palpitation	II Б	Congestion in both circles of blood circulation (pulmonary AND systemic)
IV	Symptoms are at rest and they are worse in any physical exertion	III	Structural irreversible changes in target organs (Cardiosclerosis, Pulmonary fibrosis, Liver cirrhosis, ascites, anasarca)

*SOBOE = Shortness of Breath on Exertion

Left ventricle disorders leads to pulmonary congestion (fig. 10) by two ways:

– *systolic failure* — reduced ejection fraction (*heart failure with reduced ejection fraction*). The heart can't pump with enough force to push enough blood into systemic circulation.

– *diastolic failure* — ejection fraction is normal (*heart failure with preserved ejection fraction*). The left ventricle loses its ability to relax normally due to muscle stiffness.

Complaints in case of Left Ventricle heart failure:

– shortness of breath (often on exertion, a sensitivity of 89 %);
– *orthopnea* is a dyspnea on supine position (a specificity of 89 %);
– paroxysmal nocturnal dyspnea;
– cough.

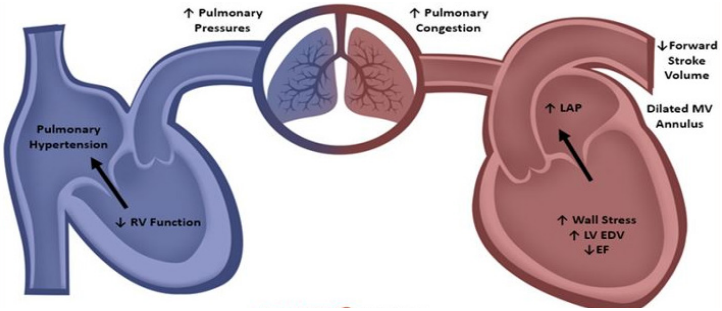


Fig. 10. Heart failure pathogenesis

Right vent ricular heart failure happens when heart's right ventricle is too weak to pump enough blood from veins to the lungs. As a result, systemic congestion develops: blood builds up in big veins, veinous pressure increases. Hydrostatic pressure pushes fluid out of the veins and into interstitial tissue. Fluid builds up in patient's liver (hepatomegaly), legs (pedal edema), abdomen (ascites), etc.

Complaints in case of Right Ventricle heart failure:

– legs edema (pale or cyanotic skin color, cold skin temperature, edema increases in evening);

– heaviness in the right hypochondrium due to liver congestion and hepatomegaly

– abdomen distension;

– decreased daily urine output (nocturia).

Since the most common cause of heart failure is damage to the left ventricle, usually the patient develops left ventricular heart failure first, than right ventricular failure, and finally biventricular heart failure develops. Objective examination in case of chronic heart failure is following (table 12).

Table 12

Objective examination in case of chronic heart failure

Method	Result
Visual examination	Orthopnea, legs edema, abdomen enlargement, skin is pale or acrocyanosis, tachypnea, jugular vein distention

End the tabl. 12

Method	Result
Palpation	Pulse is weak, asymmetric, or/and irregular, tachycardia Liver is enlarged, its surface is smooth, the edge is rounded and painful on palpation. Hepatojugular reflux is positive (jugular vein pulsation becomes visible when doctor applies firm pressure on liver)
Percussion	Relative heart dullness left border is shifted to the left, right border is shifted to the right
Auscultation	Heart sounds weaken, S3 at the apex (gallop rhythm), murmurs S2 louder at pulmonary artery Weaken vesicular breathing, dry rales (wheezing), wet rales (crackles), crepitation bilaterally at the lower lung lobes
Laboratory examination	Biochemical blood test: increased level of BNP (brain natriuretic peptide)
Instrumental examination	ECG- left ventricle hypertrophy, arrhythmia, conduction block or other abnormality (normal ECG is almost impossible) Pulse oximetry — low SpO_2 Echocardiogram — abnormal heart valves, low myocardial contractility (↓EF), high pressure in Pulmonary artery
	Chest X-ray — congestion, pleural effusion, increased cardio-thoracic ratio

ARRHYTHMIAS (HEART RHYTHM DISORDERS)

Any deviations from the normal rhythm of the heart are called arrhythmias. Cardiac arrhythmias are common in many organic and functional disorders of circulatory system. Arrhythmias are often a manifestation of structural heart disease but may also occur because of abnormal conduction or depolarization in an otherwise healthy heart due to other disorders (endocrine diseases, infection, electrolyte imbalances, etc.).

Common clinical symptoms of arrhythmias

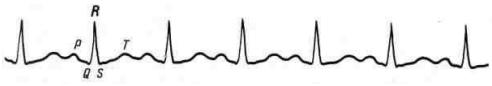
Complains: palpitation, abnormally strong or forceful heartbeats, an irregular heartbeat, chest pain, change in blood pressure, difficulty breathing, dizziness, fainting, anxiety.

Palpitation (awareness of the heartbeat) due to an arrhythmia may be accompanied by weakness, dyspnea, or light-headedness. *Hemodynamic disorders* are manifested by dizziness, syncope, arterial hypotension, dyspnea, acute heart failure. *Changes of rate and regularity of cardiac rhythm* are detected by pulse examination, heart auscultation.

Sinus tachycardia and sinus bradycardia are very common (table 13).

Table 13

Sinus tachycardia and bradycardia ECG signs

Sinus tachycardia	
	- Heart rate is 91–160/min - Sinus rhythm (normal P wave before each QRS complex)
Sinus bradycardia	
	- Heart rate is 59–40/min. - Sinus rhythm (normal P wave before each QRS complex)

Extrasystolic arrhythmia

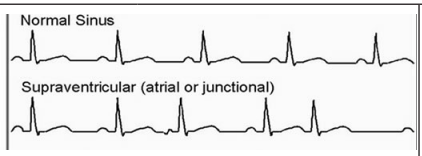
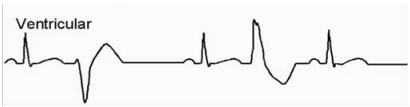
Additional (heterotopic or ectopic) foci of excitation can arise at any site of the conduction system (in the atria, ventricles, atrioventricular region). They can cause *premature contraction* of the heart before termination of the normal diastolic pause. This premature contraction is called *extrasystole*, and the disorder of the cardiac rhythm is called *extrasystolic arrhythmia* (table 14). Complaints in case of premature contraction are palpitations, an accelerated, skipped or irregular heartbeat.

Table 14

Extrasystolic arrhythmia characteristic

Pulse	Extrasystole has weaker pulse wave and a subsequent long pause. If extrasystole follows immediately a regular contraction, the left ventricle may be poorly filled with blood and the pressure inside it may be so small that the aortic valve would not open during the extrasystolic contraction and the blood will not be ejected into the aorta. The pulse wave on the radial artery will not be then detectable (missing pulse).
Auscultation of the heart	Premature contraction has specific loud first sound (due to a small diastolic filling of the ventricles)
ECG characteristics of all extrasystoles	1. Premature appearance of the ORS complex; 2. Elongated pause between the extrasystolic and subsequent normal contraction; 3. Compensatory pause — the sum of pre-extrasystolic and post-extrasystolic intervals. Complete compensatory pause equals to 2 normal R-R intervals. Incomplete compensatory pause is lesser than 2 normal R-R intervals.

End the tabl. 14

<table>
<tr><td colspan="2">1. Premature atrial contraction (atrial extrasystole)</td></tr>
<tr><td> 
</td><td>– premature extra heart beat with the P wave and the following QRS complex;
– deformation of the P wave;
– premature QRS complex is unchanged, similar in shape to normal QRS complexes of sinus origin;
– incomplete compensatory pause</td></tr>
<tr><td colspan="2">2. Premature ventricular contraction (ventricular extrasystole)</td></tr>
<tr><td> 
</td><td>– absence of a P wave before a premature contraction;
– premature ventricular contraction has deformed and prolonged QRS complex;
– the ST segment and the T wave of premature ventricular contraction are discordant to the direction of the main wave of the QRS complex;
– complete compensatory pause.</td></tr>
<tr><td>Left-ventricular extrasystole</td><td>high R wave in III standard lead and the deep S wave in I lead; high R wave in the right chest leads and a broad or deep S wave in the left chest leads</td></tr>
<tr><td>Right-ventricular extrasystole</td><td>high R wave in I lead, and a deep S wave in the III. deep S wave is recorded in the right chest leads, and a high S wave in the left chest leads</td></tr>
</table>

Atrial fibrillation, atrial flutter and ventricular fibrillation

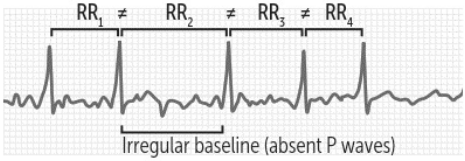
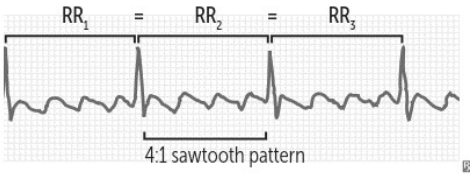
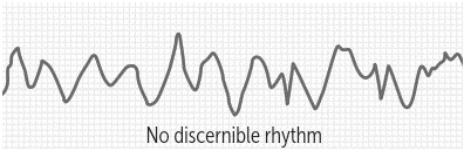
Atrial fibrillation and atrial flutter have similar pattern. In atrial fibrillation, the atria beat irregular. In atrial flutter, the atria beat regular, but faster than usual and more often than the ventricles (in both cases not all impulses can go from atria though AV nodule to ventricles) (table 15).

Complaints in case of atrial fibrillation and atrial flutter are the following: palpitations, fatigue, exertional dyspnea, dizziness, pre-syncope or syncope.

Ventricular fibrillation is considered the most serious cardiac rhythm disturbance. Ventricular fibrillation leads to sudden c ardiac arrest, which requires immediate medical attention. Signs of cardiac arrest include: loss of consciousness, absence of pulse and breathing.

Table 15

Atrial fibrillation, atrial flutter and ventricular fibrillation characteristic

Atrial fibrillation

– P-wave is absent in all leads; – The irregular wave «f» has various shape and amplitude, a frequency of 350–600 per minute; – f-wave is better visible in V1, V2, II, III and aVF leads; – QRS-complex is irregular (all R – R intervals are different); – QRS complex usually has a normal unchanged shape.
Atrial flutter

– The atrial «F» — waves are frequent (200–500 per minute), regular, similar to each other with special sawtooth shape (leads II, III, aVF, V1, V2); – In most cases, regular ventricular rhythm with the regular R – R intervals – QRS-complex has a normal shape; – each QRS-complex is preceded by a certain (often constant) number of «F» waves (2: 1, 3: 1, 4: 1, etc.)
Ventricular fibrillation

– Chaotic irregular waves of varying amplitude; – No identifiable P waves, QRS complexes, or T waves; – Rate 150 to 500 per minute; – Ventricular fibrillation is the main cause of sudden death

Conduction disorders (heart blocks)

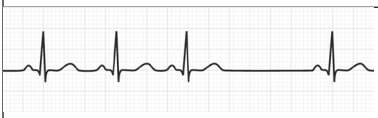
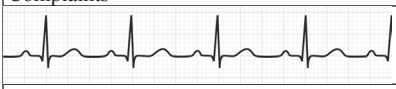
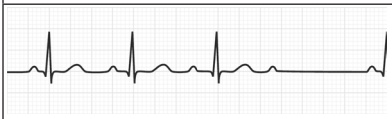
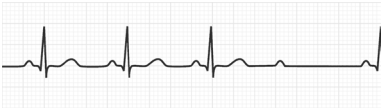
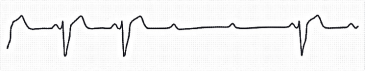
Heart blocks are delayed conduction or complete absence of conduction in some department of cardiac conduction system.

Most important conduction disorders are the following (table 16):

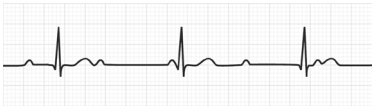
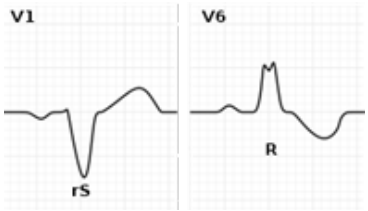
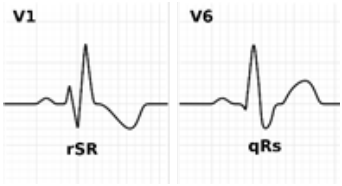
1. Sinoatrial block;
2. Atrioventricular (AV) block;
3. His bundle branch blocks.

Table 16

Conduction disorders (heart blocks) characteristics

Sinoatrial block	
Complaints	Periodic missing of the heart beat and pulse beat.
	ECG signs: -periodic missing of the heart complex (PQRST) in the presence of a regular sinus rhythm; -the length of diastole doubles.
Atrioventricular (AV) block	
1st Degree AV Block	
Complaints	Absent
	- PR interval is fixed > 0.20 sec; - all normal P waves are followed by QRS complexes.
2nd Degree AV Block	
Complaints	Periodic missing of the heart beat and pulse beat. Dizziness, presyncope and syncope in case of several missed heart beats.
	Type 1 (Mobitz 1) - PR interval gets longer and longer until a nonconducted P wave occurs (one beat drops) - Wenckebach phenomenon.
	Type 2 (Mobitz 2) - PR interval remains constant (normal or prolonged). - Beats are intermittently nonconducted and QRS complexes dropped - Block may present as a single nonconducted P wave or a repetitive pattern of nonconduction (2:1, 3:1, etc.)
	High-grade AV block - Two or more consecutive blocked P waves.

End the tabl. 16

3d Degree AV Block (Complete)	
Complaints	Dizziness, fatigue, confusion, syncope, chest pressure or pain, dyspnea
	- No atrial impulses conduct to the ventricle and P-waves have no relation to the QRS complexes (AV dissociation); - The atrial rate (PP) is faster than the independent ventricular rate (RR); - P wave may occur on any part of the curve.
His bundle branch block	
Complaints	Absent
ECG signs	- P wave does not change; - ventricles contract rhythmically by the impulse from the sinus node; - QRS complexes are markedly prolonged ≥ 0.12 s; - The shape of the ventricular complexes depends on the particular bundle branch which is blocked.
Left bundle branch block	Complete QRS > 0,12 sec.
	- Wide, notched R wave in leads I, aVL, V5 and V6. - QRS complex has the shape QS or rS in leads V1, V2, III, aVF (S wave is wide and notched). - Absence of Q wave in leads I, V5 and V6. - ST segment and T wave are generally opposite in direction to QRS. - The mean QRS axis turns to the left (sometimes to the right).
Right bundle branch block	Incomplete QRS 0,11–0,12 sec. Complete QRS > 0,12 sec.
	- The QRS complex appears as the letter “M” in the leads V1-V2 (rSR, rsR,). The second R-wave is always larger than the first R-wave. - S-wave is wide, notched in the leads V5, V6, aVL. - V1–V2 leads show downsloping ST-segments and inverted T-waves.

ACUTE CIRCULATORY FAILURE

Acute circulatory failure is a pathologic state developed due to decreased vessel's tone and arterial hypotension. It can be perceived like syncope, collapse or shock.

Syncope (fainting or passing out) is sudden short-term loss of consciousness caused by insufficient blood flow to the brain. Syncope can occur as a result of stress, severe pain, cardiac arrhythmias, structural heart disease (heart valve diseases, ischemic heart diseases, cardiomyopathy), stimulation of carotid sinus, etc.

Fainting lasts from a few seconds to one minute, rarely longer. On examination, the patient's skin is pale, the pulse is rare and faint, blood pressure is low. Heart auscultation can show tachycardia, irregular heartbeats (in case of arrhythmias) or murmurs (in case of heart valve diseases).

Collapse and shock are the forms of acute vascular insufficiency, based on cardiac output decrease. In such a case the cardiac output is not sufficient for normal tissue supply. The causes of collapse and shock are the following: severe bleeding, myocardial infarction, heart rhythm disorders, severe infection, poisoning, trauma.

Collapse is a moderate form of vascular insufficiency (hypotension is the main symptom). Consciousness is always present in case of collapse, but can be confused. Skin is pale and cold, acrocyanosis can present. Blood pressure is low, pulse is frequent and weak.

Shock is the most severe form of vascular insufficiency. Shock leads to the acute metabolic disorders, microcirculation, the organs and systems disturbance. With any type of shock severe hypotension is present; the patient's skin is moist, cold and cyanotic, sometimes with a «marble» pattern. The patient's consciousness is inhibited, but is present, pulse is frequent and thready, breathing is shallow. Heart sounds are weak in auscultation, tachycardia, irregular heartbeats, murmurs or «gallop» can be heard.

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ОГЛАВЛЕНИЕ

Учебное издание

Доценко Эдуард Анатольевич
Шолкова Мария Владимировна
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КЛИНИЧЕСКИЕ СИНДРОМЫ ПРИ ЗАБОЛЕВАНИЯХ СЕРДЕЧНО-СОСУДИСТОЙ СИСТЕМЫ

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Ответственный за выпуск Э. А. Доценко
Переводчики М. В. Шолкова, Ю. В. Репина
Компьютерная вёрстка А. В. Янушкевич

Подписано в печать 13.10.22. Формат 60×84/16. Бумага писчая «Xerox office».
Ризография. Гарнитура «Times».
Усл. печ. л. 1,63. Уч.-изд. л. 1,2. Тираж 99 экз. Заказ 464.
Издатель и полиграфическое исполнение: учреждение образования
«Белорусский государственный медицинский университет».
Свидетельство о государственной регистрации издателя, изготовителя,
распространителя печатных изданий № 1/187 от 18.02.2014.
Ул. Ленинградская, 6, 220006, Минск.

ISBN 978-985-21-1141-6